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RESEARCH ARTICLE

Separating the effects of habitat area, fragmentation and matrix resistance on genetic differentiation in complex landscapes

Samuel A. Cushman · Andrew Shirk · Erin L. Landguth

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Abstract Little is known about how variation in landscape mosaics affects genetic differentiation. The goal of this paper is to quantify the relative importance of habitat area and configuration, as well as the contrast in resistance between habitat and non-habitat, on genetic differentiation. We hypothesized that habitat configuration would be more influential than habitat area in influencing genetic differentiation. Population size is positively related to habitat area, and therefore habitat area should affect genetic drift, but not gene flow. In contrast, differential rates and patterns of gene flow across a landscape should be related to habitat configuration. Using spatially explicit, individual-based simulation modeling, we found that habitat configuration had stronger relationships with genetic differentiation than did habitat area, but there was a high degree of confounding between the effects of habitat area and configuration. We evaluated the predictive ability of six widely used landscape metrics and found that patch cohesion and correlation length of habitat are among the strongest individual predictors of genetic differentiation. Correlation length, patch density and clumpy are the most parsimonious set of variables to predict the magnitude of genetic differentiation in complex landscapes.

Keywords Landscape genetics · Area · Configuration · Fragmentation · Limiting factors · CDPOP · Simulation · Thresholds

S. A. Cushman (✉)
U.S. Forest Service, Rocky Mountain Research Station, 2500 S. Pine Knoll Drive, Flagstaff, AZ 86001, USA
e-mail: scushman@fs.fed.us

A. Shirk
JISAO Climate Impacts Group, University of Washington, Seattle, WA, USA
e-mail: ashirk@u.washington.edu

E. L. Landguth
Division of Biological Sciences, University of Montana, Missoula, MT, USA
e-mail: erin.landguth@mso.umt.edu

Introduction

Quantifying the effects of habitat area and fragmentation on ecological processes has emerged as one of the central questions in landscape ecology. Population connectivity is central to many ecological processes and conservation goals. For example connectivity is a key factor affecting regional viability of animal populations (Hanski and Ovaskainen 2000; Flather and Bevers 2002; Cushman 2006). Habitat loss and fragmentation affect population density (Wiegand et al. 1999, 2005; Revilla and Wiegand 2008) and distribution (Fahrig and Merriam 1985; Fahrig and Paloheimo 1988; Hanski and Ovaskainen 2000), and can decrease dispersal (Gibbs 1998), reduce genetic diversity (Reh and Seitz 1990; Keyghobadi 2007), and

increase mortality (Fahrig et al. 1995). Populations may not be recolonized following local extinction if immigration is prevented (Brown and Kodric-Brown 1977; Harrison 1991; Semlitsch and Bodie 1998). Thus, the ability of individual animals to move across landscapes is critical for maintaining populations (Fahrig 2003; Cushman 2006; Lindenmayer and Fischer 2007).

Considerable debate has recently focused on the relative importance of habitat area versus habitat configuration in driving population processes. Empirical and simulation studies generally suggested that the effects of fragmentation on species occurrence and extinction risk are generally weaker than the effects of habitat loss (Fahrig 2003). Theoretical studies have shown that landscape composition is more important than configuration in determining landscape occupancy, and that configuration becomes important only at low levels of habitat area (With and Crist 1995; Fahrig 1997, 1998; Hill and Caswell 1999; With and King 1999; Fahrig 2001, 2002; Flather and Bevers 2002). Results of empirical studies, mostly on birds, have been generally consistent with these theoretical findings (Andren 1994; McGarigal and McComb 1995; Trzcinski et al. 1999; Villard et al. 1999; Cushman and McGarigal 2002; Schmiegelow and Monkkonen 2002; Cushman and McGarigal 2004).

Some studies have modeled movement in an individual-based, spatially explicit framework to evaluate fragmentation thresholds and connectivity of landscape mosaics (e.g. Schumaker 1996; With and King 1999). However, few studies have addressed the effects of habitat loss and fragmentation on gene flow in complex landscapes. Ezard and Travis (2006) quantified thresholds for fixation time of selectively neutral genotypes by genetic drift in complex landscapes. They found that fixation time was determined by habitat shape and spatial correlation of habitat loss. Bruggeman et al. (2010) used simulation modeling to quantify both the influence of patch size and isolation on abundance, effective population size and *F*st in red-cockaded woodpecker. Their results suggest that population genetic structure is more strongly affected by habitat fragmentation than habitat patch size.

There are theoretical reasons to expect that processes governing extinction risk should be more related to habitat area than configuration, because population size is positively related to habitat area, and extinction risk is highly related to population size. In addition, patterns of occurrence and abundance in mobile species should be more highly related to habitat area than configuration because mobile animals can integrate a fractured landscape such that it behaves as functionally connected habitat. However, this does not suggest that habitat area will be more important than configuration in driving genetic differentiation. Changing habitat area without changing habitat configuration should have no effect on spatial patterns of gene flow. Gene flow is driven by patterns of mating and dispersal, which in homogenous landscapes are driven by isotropic isolation by distance processes (Landguth and Cushman 2010). Non-isotropic spatial genetic structure will only emerge when landscape structure differentially affects gene flow (Ezard and Travis 2006; Landguth et al. 2010). For example, Short Bull et al. (2011) found that landscape features such as forest cover, roads and elevation have strong influences on population connectivity of American black bear (*Ursus americanus*), but only affect genetic differentiation when their high heterogeneity limits gene flow across the landscape. Therefore, it is important to directly investigate the mechanisms that drive population connectivity (Cushman 2006; Lindenmayer and Fischer 2007), and quantify the relative influence of changes in habitat area and configuration on genetic differentiation.

Simulation modeling provides explicit control over pattern–process relationships (Epperson et al. 2010). This enables rigorous attribution of the causes of genetic differentiation, evaluation of factor complexes that would be impossible to directly investigate in the field (Balkenhol et al. 2009; Epperson et al. 2010; Segelbacher et al. 2010). Specifically, simulation provides a means to rigorously evaluate the relative effects of habitat area and configuration on genetic differentiation, which is only possible when habitat area can be varied independently of configuration (McGarigal and Cushman 2002; Fahrig 2003).

In this paper we use spatially explicit, individual-based simulations of genetic exchange to evaluate the relative effects of habitat area and fragmentation on genetic differentiation in complex landscapes. Our major goals were to quantify the relative importance of habitat area and fragmentation on landscape genetic patterns and evaluate a range of fragmentation metrics in terms of their influence on genetic differentiation. We expected that habitat area and fragmentation would both affect the apparent strength of landscape

genetic patterns, but that variation in habitat configuration would have larger effects than habitat area.

Methods

Factorial modeling

We designed a factorial modeling experiment to explore how variation in habitat area, fragmentation and relative resistance of habitat and non-habitat affect the strength of landscape genetic relationships. We used the neutral landscape model QRULE (Gardner 1999) to produce simulated binary landscape maps (habitat vs. non-habitat) which varied in habitat area and fragmentation (Fig. 1). All maps were 512 × 512 pixels. QRULE controls fragmentation through the H parameter, which affects the aggregation of pixels into homogeneous patches. We varied habitat area across five levels (15%, 35%, 55%, 75%, 95%), and H across five levels (0.2, 0.4, 0.6, 0.8, 1.0). An H parameter value of 0 indicates zero fractal aggregation, while a value of 1 indicates extreme aggregation. We varied the ratio of resistance of non-habitat relative to habitat across five levels (1.5, 2, 4, 8, 16) to investigate how increasing resistance of the matrix affects the strength and detectability of landscape genetics relationships in interaction with habitat area and fragmentation. Finally, we produced five replicates of this factorial, producing a total of 625 landscapes for analysis.

Selection of landscape fragmentation metrics

We selected a suite of landscape metrics to measure habitat fragmentation in the simulated maps based on past work which assessed the strength and functional shape of relationship between a large number of landscape metrics and habitat proportion (P) and the H level in QRULE landscapes (Neel et al. 2004). Also, several landscape configuration metrics have previously been shown to be sensitive indicators of the effects of landscape structure on spatial population

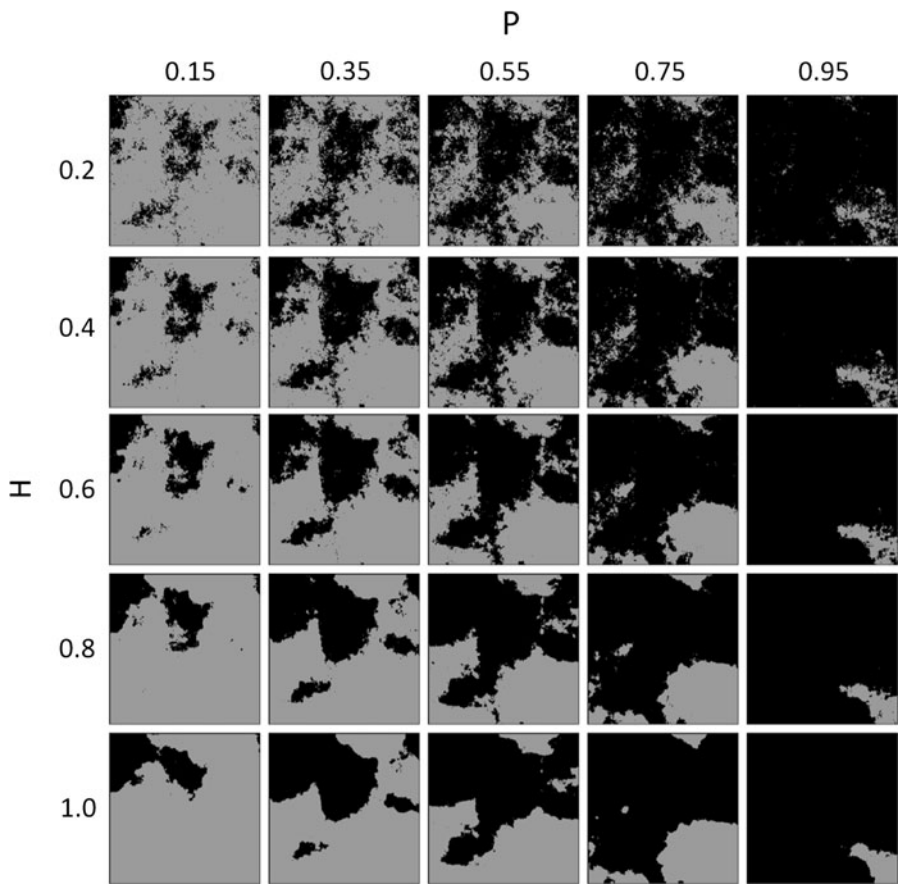

Fig. 1 We evaluated a three-way factorial of habitat area (P), habitat fragmentation (H) and contrast in landscape resistance between habitat and non-habitat. This figure shows one replicate of the full combination of P and H. For each combination, we evaluated five levels of relative landscape resistance (1.5, 2, 4, 8, 16), and produced five replicates of each combination of habitat area and fragmentation using different random number seeds

processes (e.g. patch cohesion, Schumaker 1996) and gene flow (correlation length, Cushman et al. 2010; Short Bull et al. 2011). Based on these considerations, we chose patch density (PD), correlation length (GYRATE_AM), the clumpy index of class aggregation (CLUMPY), patch cohesion (Cohesion), and aggregation index (AI) (McGarigal et al. 2002). Correlation length quantifies the extensiveness of patches in spanning the landscape in terms of the average distance an organism could travel in a random direction and remain in habitat when dropped randomly in a habitat patch. Patch density quantifies the spatial density of disjunct habitat patches. The clumpy index (McGarigal et al. 2002) measures class aggregation independently of class area (Neel et al. 2004). As such it is a useful metric to quantify the relative importance of habitat area and fragmentation (Cushman et al. 2008). Patch cohesion and aggregation index are metrics that quantify class aggregation. The aggregation index is commonly used in fragmentation studies, and patch cohesion was shown by Schumaker (1996) to be a strong indicator of habitat fragmentation effects on population connectivity. Percentage of the landscape of habitat (PLAND) is the most universal measure of landscape composition, and is recommended in all landscape pattern analyses (Cushman et al. 2008), and we included it to enable comparison of relative magnitude of area versus configuration effects. All metrics were calculated using FRAGSTATS (McGarigal et al. 2002).

Landscape genetic simulation with CDPOP

We used CDPOP version 0.84 (Landguth and Cushman 2010) to simulate the processes of mating and dispersal as functions of the spatial patterns of habitat and non-habitat on these 625 simulated landscapes. CDPOP is an individual-based, spatially explicit, landscape genetic model that simulates birth, death, mating and dispersal of individuals in complex landscapes as probabilistic functions of movement cost among them. CDPOP models genetic exchange for a given resistance surface and n individuals as functions of individual-based movement through mating and dispersal, vital dynamics, and mutation. The model represents landscape structure as a resistance surface whose value represents the step-wise cost of crossing each location. Mating and dispersal are modeled as probabilistic functions of cumulative cost across these resistance surfaces. It provides a framework for simulating the spatial genetic resulting from specified landscape resistance governing movement.

In each of the 625 landscape maps, we randomly placed 500 individuals in habitat pixels. We simulated gene flow among these locations for 500 non-overlapping generations to ensure genetic equilibrium. Each individual's genetic data consisted of ten loci, each initialized with 10 alleles randomly assigned within each locus. We used an inverse square mating and dispersal probability function, with maximum dispersal cost-weighted distance of 7,680 m (the diameter of the simulated landscapes) in ideal habitat (i.e. a resistance value of one). The range of relative resistance of non-habitat represents different dispersal distances through non-habitat, (e.g. 5,130 m for r1.5; 3,840 m for r2; 1,920 m for r4; 960 m for r8, and 480 m for r16). Reproduction was sexual with non-overlapping generations, and the number of offspring was based on a Poisson distribution with a mean of four. We ran ten replicate runs in CDPOP to assess stochastic variability, producing 6,250 CDPOP simulations.

Mantel tests

CDPOP output included matrices of pairwise genetic distances between all 500 simulated individuals based on the proportion of shared alleles at generation 500 (Bowcock et al. 1994). We calculated a matrix of pairwise effective landscape distances between all individuals using the COSTDISTANCE function in ArcGIS (ESRI 1999–2008). To assess the relationship between genetic and landscape distance matrices, we used partial Mantel tests (Mantel 1967), implemented in the ECODIST package in *R* (Goslee and Urban 2007). We calculated partial Mantel *r* (Smouse et al. 1986; removing the effect of geographical distance) for all 6,250 simulated populations at generation 500 and assessed statistical significance based on 9,999 permutations.

Variance partitioning

We used variance partitioning to quantify the relative effects of habitat amount versus habitat configuration for each of the five landscape configuration metrics (Cushman and McGarigal 2002). Variance

partitioning quantifies the independent and joint effects of multiple explanatory variables (Borcard et al. 1992). In our case we are partitioning the variance in partial Mantel *r* values that is explained by habitat amount alone, the configuration metric alone, and that is jointly and simultaneously explained by both habitat amount and the configuration metric. The variance partitioning is accomplished by computing simple and partial Pearson correlations between partial Mantel *r*, habitat amount and each configuration metric (Borcard et al. 1992). We also wished to evaluate if the relative effect size of habitat amount and configuration changed with different levels of relative matrix resistance. Accordingly, we computed variance partitioning for each of the five landscape configuration metrics, at each of the five relative matrix resistance values (r1.5, r2, r4, r8, r16).

Mantel *r* and partial Mantel *r* as functions of habitat area and fragmentation

We used generalized linear models to predict simple and partial Mantel *r* values as functions of habitat area, fragmentation and relative landscape resistance (R Development Core Team 2009). We screened variables for inter-correlation, given the sensitivity of regression analysis to colinearity of predictor variables. Correlation length (GYRATE_AM), aggregation index (AI) and patch cohesion (COHESION) were highly correlated with percentage of the landscape occupied by habitat (PLAND). We proposed a suite of candidate models that excluded pairs of highly inter-correlated variables.

We adopted an information theoretic approach (Burnham and Anderson 2002). A priori, we identified 25 candidate models (Table 2). The candidate model pool includes all variables individually, and all combinations of variables that are not highly inter-correlated (less than Pearson *r* 0.7). We used AIC to rank models and used model averaging to produce final models predicting partial Mantel *r* as a function of habitat area, fragmentation and relative landscape resistance. We estimated variable importance in three ways. First, we calculated the AIC importance weight by summing the AIC weights of all models including each variable. Second, Smith et al. (2009) suggested that standardized regression coefficients are among the best measures of effects size in studies comparing the relative effects of habitat area and configuration. Additionally, we calculated the predicted change in the dependent variable as each variable changed from the 10th to the 90th percentile of the distribution of the simulated landscapes, while holding all other independent variables constant at their medians.

Results

Variance partitioning

We produced 25 partitionings of variance in partial Mantel *r* value explained by habitat amount and each of the configuration metrics, across the five levels of relative matrix resistance (Table 1). At the lowest level of relative matrix resistance (r1.5), neither habitat amount nor configuration explained any variance in partial Mantel *r* (Table 1; Fig. 2). The total amount of variance explained by habitat amount and each configuration metric increased with increasing relative matrix resistance up to resistance r8, before decreasing at resistance r16 (Table 1; Fig. 2). The independent influence of patch cohesion was greater than habitat amount at resistance levels r2, r4 and r8 (Fig. 2a). At resistance level r16 the independent effects of habitat area were greater than those of patch cohesion. A similar pattern was seen for aggregation index (Fig. 2b).There was no shared explained variance between CLUMPY and PLAND at any level of relative matrix resistance, and habitat amount always explained a larger portion of variance in partial Mantel *r* than CLUMPY (Fig. 2c).There was very little independent influence of habitat amount after removing the effect of habitat correlation length across all levels of relative matrix resistance (Fig. 2d). In contrast, across all levels of matrix resistance, habitat correlation length had substantial explanatory power independent of habitat area (Fig. 2d).

Univariate regression

Habitat correlation length and patch cohesion were tied as the most important predictors of partial Mantel *r* based on the magnitude of standardized regression coefficients (Table 2). Conversely, Clumpy was the least important variable, and patch density the second least important, based on this criteria. Based on change in predicted Mantel *r* from the 10th to the 100th percentile, patch cohesion had the largest effect size

Table 1 Variance partitioning results for separating the effects of habitat area from each of the configuration metrics, across the five levels of relative matrix resistance (1.5×, 2×, 4×, 8×, 16×)

	r1.5		r2		r4		r8		r16	
	% total	% exp	% total	% exp	% total	% exp	% total	% exp	% total	% exp
p	0.003	0.710	0.011	0.047	0.007	0.030	0.012	0.031	0.066	0.265
cohesion	0.001	0.290	0.070	0.317	0.057	0.254	0.143	0.386	0.023	0.092
P*cohesion	0.000	0.000	0.141	0.635	0.160	0.717	0.215	0.582	0.161	0.643
p	0.002	0.941	0.160	0.850	0.176	0.633	0.242	0.841	0.228	0.991
clumpy	0.000	0.036	0.028	0.150	0.102	0.367	0.046	0.159	0.002	0.009
p*clumpy	0.000	0.023	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
p	0.005	0.589	0.001	0.003	0.009	0.038	0.005	0.015	0.001	0.003
gyrate_am	0.004	0.411	0.035	0.189	0.074	0.308	0.078	0.257	0.032	0.123
p*gyrate_am	0.000	0.000	0.151	0.808	0.157	0.654	0.222	0.728	0.226	0.874
p	0.002	0.930	0.107	0.629	0.118	0.632	0.162	0.610	0.195	0.856
pd	0.000	0.011	0.019	0.111	0.021	0.113	0.040	0.148	0.001	0.004
p*pd	0.000	0.058	0.044	0.260	0.048	0.255	0.064	0.242	0.032	0.139
p	0.002	0.913	0.049	0.226	0.057	0.243	0.080	0.240	0.146	0.624
ai	0.000	0.087	0.064	0.297	0.066	0.285	0.107	0.321	0.006	0.028
p*ai	0.000	0.000	0.103	0.477	0.110	0.472	0.147	0.439	0.081	0.348

% total is the proportion of variance in partial Mantel *r* values that is explainable by that variance component. % exp is the proportion of the variance in partial Mantel *r* values explained by all three components that is explained by that variance component

p amount of variance in partial Mantel *r* values independently explained by habitat amount alone, *"metric"* amount of variance in partial Mantel *r* values independently explained by that configuration metric alone, *p*"metric"* amount of variance in partial Mantel *r* values jointly and simultaneously explained by both habitat amount and that metric, *r1.5* matrix 1.5× as resistant as habitat, *r2* matrix 2× as resistant as habitat, *r4* matrix 4× as resistant as habitat, *r8* matrix 8× as resistant as habitat, *r16* matrix 16× as resistant as habitat

(0.208; Table 2), followed by aggregation index (AI), and correlation length of habitat (GYRATE_AM). The clumpy index of aggregation was the least powerful predictor, with a decrease in equilibrium partial Mantel *r* of 0.032 as CLUMPY changed from the 10th to the 100th percentile. The percentage of habitat in the landscape was the third most influential variable based on standardized regression coefficients, and fourth most influential based on effect size, with the predicted equilibrium partial Mantel *r* decreasing by 0.133 as habitat increased from 10 to 100% of the landscape.

Multivariate regression

Three models had non-zero AIC weights (Table 3). The final averaged model includes correlation length (GYRATE_AM), clumpy, patch density and relative resistance value (Table 4). Correlation length was the most influential variable, based on all three measures of variable importance (Table 4). Relative landscape resistance of habitat compared with non-habitat was the second most important variable. Based on the magnitude of the standardized regression coefficient, correlation length had a 42% larger effect size than relative landscape resistance, while it had 19.8% larger effects size when calculated based on change in predicted Mantel *r* from 10th to 90th percentile holding the other variables constant at their medians. Patch density and clumpy had much weaker effects than either correlation length or relative landscape resistance. For example, based on standardized regression coefficients, correlation length of habitat had 8.3 times greater influence on equilibrium Mantel *r* than patch density, and 16.8 times greater influence than CLUMPY. The relative importance of these two variables was similar based on the percentage change in partial Mantel *r* from the 10th to the 90th percentile, with correlation length predicted to have 3.13 times and 4.76 times the influence of patch density and CLUMPY, respectively.

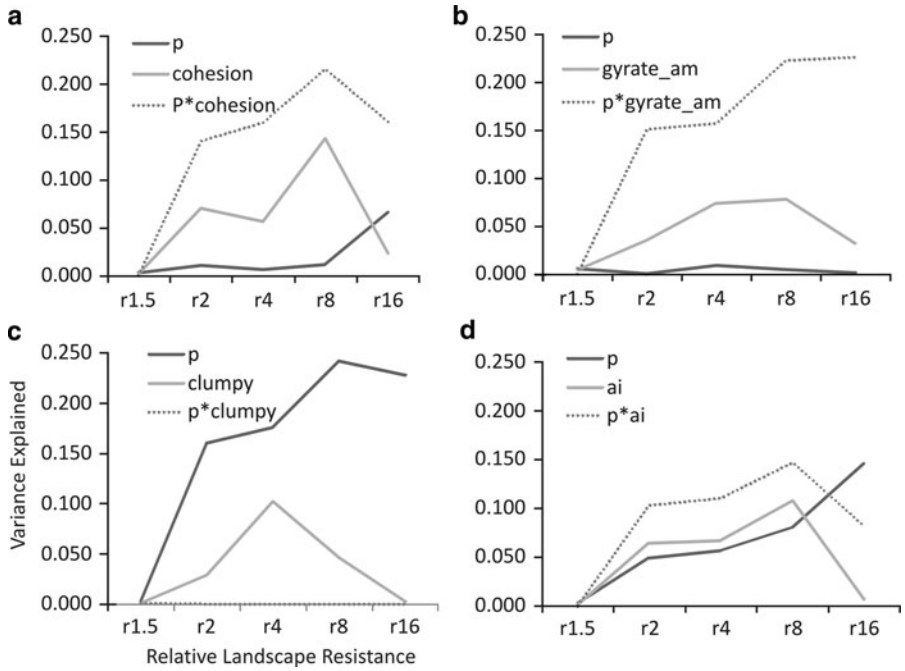

Fig. 2 Change in amount of variance in each of the three variance partitioning components resulting from partitioning the variance in partial Mantel *r* explained by habitat area (p), each individual configuration metric (**a** patch cohesion [cohesion]; **b** correlation length [gyrate_am]; **c** clumpy index of aggregation [clumpy]; **d** aggregation index [ai]), across the five levels of relative matrix resistance (matrix 1.5× as resistant as habitat [r1.5]; matrix 2× as resistant as habitat [r2]; matrix 4× as resistant as habitat [r4]; matrix 8× as resistant as habitat [r8]; matrix 16× as resistant as habitat [r16]. *P* amount of variance in partial Mantel *r* values independently explained by habitat amount alone, *"metric"* amount of variance in partial Mantel *r* values independently explained by that configuration metric alone, *p*"metric"* amount of variance in partial Mantel *r* values jointly and simultaneously explained by both habitat amount and that metric

Table 2 Univariate effect sizes for all independent variables

	PLAND	GYRATE_AM	PD	CLUMPY	COHESION	AI
Standardized Coefficient	−0.047	−0.052	0.027	−0.01	−0.052	−0.042
Change in predicted equilibrium Mantel *r*	−0.133	−0.138	−0.082	−0.032	−0.208	−0.149

Three measures of effect size (AIC variable importance, standardized regression coefficient, and change in predicted equilibrium Mantel *r* when the predictor variable changes from the 10th to the 90th percentile of simulated landscapes) for univariate regression models predicting equilibrium Mantel *r* as functions of each of the six independent variables alone

Discussion

There are theoretical reasons to expect habitat configuration to be more important than habitat area in driving genetic differentiation. Within a homogeneous habitat patch gene flow will be governed by isotropic isolation by distance processes (Landguth et al. 2010). Under isotropic isolation by distance genetic differentiation between individuals will increase with distance. The rate at which genetic distance increases and the variance in genetic distance among individuals will be a function of the dispersal ability of the species. The range and functional shape of the dispersal function will drive the range of significant genetic autocorrelation and the rate at which genetic differentiation increases with distance (Landguth and Cushman 2010). However, simply changing the extent of the patch will not change spatial genetic structure. Increasing the size of a homogeneous habitat patch in which genetic differentiation is governed by isolation by distance will have little effect on genetic differentiation, because it does not change the rate of genetic

Table 3 AIC, delta AIC and AIC weights for the 25 candidate models predicting partial Mantel *r* as functions of relative resistance of habitat (R) and non-habitat, habitat area (pland), the clumpy index (clumpy), patch density (PD), correlation length of habitat (GYRATE_AM), patch cohesion (cohesion) and aggregation index (AI)

Model	AIC	delta AIC	w
GYRATE_AM + clumpy + PD + R	−9304.8	0	0.52
GYRATE_AM + PD + R	−9304	0.8	0.35
GYRATE_AM + clumpy + R	−9302.1	2.7	0.13
cohesion + clumpy + PD + R	−9289.3	15.5	0.00
cohesion + clumpy + R	−9286.3	18.5	0.00
GYRATE_AM + R	−9282	22.8	0.00
cohesion + R	−9266.7	38.1	0.00
AI + clumpy + PD + R	−9266.6	38.2	0.00
cohesion + PD + R	−9266.4	38.4	0.00
AI + PD + R	−9170.2	134.6	0.00
pland + clumpy + R	−9145.6	159.2	0.00
pland + PD + R	−9145.6	159.2	0.00
pland + R	−9145.6	159.2	0.00
pland + clumpy + pd + R	−9145.5	159.3	0.00
AI + clumpy + R	−9088	216.8	0.00
AI + R	−8872.9	431.9	0.00
GYRATE_AM	−8740	564.8	0.00
cohesion	−8726	578.8	0.00
pland	−8548.5	756.3	0.00
AI + R	−8440.2	864.6	0.00
AI	−8364	940.8	0.00
clumpy + R	−8178.9	1125.9	0.00
R	−8140.2	1164.6	0.00
PD	−7964.2	1340.6	0.00
clumpy	−7721.7	1583.1	0.00

Table 4 Model averaged parameter estimates for prediction of partial Mantel *r* values as functions of landscape fragmentation, and two measures of variable importance

	Intercept	Correlation length	Patch density	Clumpy	R
Coefficient	0.048522	−0.04925	−0.00276	0.005274	0.034698
Standard error	0.001453	0.001617	0.001288	0.001745	0.001453
AIC variable importance		1.00	0.87	0.65	1.00
Change in Mantel *r* from 10th to 100th percentile		−0.10856	0.018834	0.026237	0.0906

The variables included in the model are: correlation length of habitat (GYRATE_AM), patch density (PD), clumpy index (CLUMPY), and relative resistance of matrix compared to habitat (R). Change in estimated Mantel *r* given change from 10th to 90th percentile of each variable, holding the other variables constant at their medians. AIC variable importance is the sum of AIC weights of models including that variable

differentiation with distance or the spatial range of significant autocorrelation. In contrast, the pattern of habitat in a landscape will affect the distribution of the population and the degree of connectivity across it, resulting in differential patterns of gene flow (Ezard and Travis 2006). Changes in the configuration of the landscape will directly affect genetic differentiation because it directly affects the spatial pattern of mating and dispersal, which drives genetic differentiation. Thus, strength of spatial genetic structure in a population should be strongly influenced by the complexity of the landscape mosaic and the degree of contrast in resistance to gene flow of the different elements making up that mosaic. Thus we predicted

that there would be strong relationships between habitat configuration, landscape resistance and genetic differentiation, and weaker relationships between habitat area and genetic differentiation.

Separating the effects of habitat loss and fragmentation is difficult as habitat amount and configuration are inextricably linked (Fahrig 2003; Smith et al. 2009). Population responses to habitat loss and fragmentation are related to the percolation properties of the landscape (With and King 1997, 1999). The potential range of landscape configuration becomes truncated at both very high and very low habitat areas (Gardner et al. 1989; Neel et al. 2004). For example, it is impossible to have high habitat fragmentation in a landscape that is covered in a very high proportion of habitat, given that there are few ways to break up patches that cover nearly the entire landscape (Gardner et al. 1987; With and King 1997). Neutral landscape models (Gardner et al. 1989) coupled with individual-based genetic simulation provides a unique means to control the pattern–process relationships and isolate the relative effects of habitat area versus habitat configuration.

By simulating across a broad factorial of habitat composition and configuration, we were able to evaluate the relative importance and interaction of habitat area, habitat fragmentation and contrast in landscape resistance on effects size and power to detect relationships. The neutral models we employed (QURLE, Gardner 1999) enable control of two attributes of landscape structure, including habitat area and the fractal aggregation parameter H. However, many aspects of the configuration of the landscapes produced by QRULE are not directly a function of H and are not independent of P. In our analysis we found that levels of H poorly explained genetic differentiation, while several configuration metrics, such as correlation length, aggregation index and patch cohesion, had strong relationships with genetic differentiation. The variance partitioning also showed that these landscape configuration metrics were substantially correlated with habitat area. The relatively high confounding of habitat area and landscape configuration metrics in terms of their ability to explain genetic differentiation makes it impossible to formally separate relative influence of habitat area and fragmentation on genetic differentiation. However, the larger marginal and independent explanatory ability of single configuration metrics compared to habitat area, and the dominance of the multivariate model by configuration metrics suggests that habitat configuration is more important than habitat area in driving genetic differentiation.

Our results contrast somewhat with several other studies that evaluated the effects of landscape structure on genetic differentiation. Ezard and Travis (2006) used neutral landscape models and genetic simulation to evaluate how habitat loss and fragmentation affected time to fixation. Our analysis differs from that f Ezard and Travis (2006) in several important ways. Our goal was not to quantify global time to fixation, but rather the strength of correlation between genetic distances and cost distances among individuals distributed across complex landscapes as functions of habitat area, fragmentation and relative landscape resistance. This enabled us to evaluate the effect of landscape structure on the strength and detectability of genetic heterogeneity in the population. Bruggeman et al. (2010) used simulation modeling to quantify both the influence of patch size and patch isolation on abundance, effective population size and *F*st in red-cockaded woodpecker. Their results suggested that population genetic structure is more strongly affected by habitat fragmentation than habitat patch size. Our results confirmed that habitat configuration is more important than composition in predicting genetic differentiation.

We extended this by evaluating the predictive ability of six widely used landscape metrics. Putting our analysis directly in terms of well understood landscape metrics helps link landscape genetics directly with landscape pattern analysis and assessment of the relative effects of habitat loss and fragmentation. Showing that patch cohesion, correlation length and aggregation index are strong single predictors of genetic differentiation, and that correlation length, patch density and clumpy are the most parsimonious set of predictor variables, provides valuable guidance to scientists in selecting landscape metrics for use in landscape genetic analyses.

The clumpy index of habitat aggregation was formulated to explicitly quantify class aggregation independently from habitat amount (McGarigal et al. 2002). This theoretically would make it an ideal metric to evaluate the independent relationship between habitat configuration and genetic differentiation. It is interesting that the clumpy index of aggregation (CLUMPY) was the least powerful predictor, given

it was specifically formulated to be an unbiased measure of habitat aggregation across all amounts of habitat area (McGarigal et al. 2002; Neel et al. 2004). This suggests that the attributes of landscape configuration this metric quantifies, while independent of habitat area, are relatively unimportant in predicting gene flow in complex landscapes. In contrast patch cohesion, correlation length and aggregation index had large independent explanatory abilities after removing the effect of habitat area. Importantly, the observation that the components of variance explained by these metrics that are independent of habitat area are much larger than the components of variance explained by habitat area independent of these metrics shows that habitat the attributes of habitat configuration measured by correlation length, patch cohesion and aggregation index have substantially stronger relationships with genetic differentiation than does habitat area.

Schumaker (1996) found that patch cohesion was an effective landscape configuration metrics in predicting population connectivity in simulation of spotted owl (*Strix occidentalis*) population dynamics in fragmented landscapes. Similarly, Short Bull et al. (2011) showed that the correlation length of habitat in a landscape is a strong predictor of the strength of genetic differentiation in American black bear (*U. americanus*). Our results confirm that patch cohesion and correlation length are among the best single metrics for studies that aim to quantify functional habitat configuration relative to movement and gene flow. Our multivariate model suggests that correlation length in combination with patch density and clumpy provide the most parsimonious set of landscape metrics for predicting genetic differentiation in complex landscapes.

Habitat configuration results in genetic differentiation by creating spatially heterogeneous patterns of local gene flow (Landguth et al. 2010). Heterogeneous patterns of gene flow, in turn, are driven by differential connectivity across the landscape as functions of landscape heterogeneity and the relative cost of movement through habitat and matrix. Thus, increasing landscape complexity, in terms of correlation length, patch cohesion, or aggregation index, is strongly related to the strength of correlation between genetic differentiation and cost distance between pairs of individuals in a population.

Scope and limitations

The results of this study should be interpreted within the scope of inference enabled by the analysis. First, using neutral landscape models to produce study landscapes across combined gradients of habitat extent and fractal aggregation is critical to formally separate the effects of habitat area and fragmentation (Fahrig 2003; With and King 1999). However, the neutral model controlled only these two properties. Importantly, much of the variation in landscape configuration in the simulated landscapes varied independently of H and was in part dependent on P. Thus, even using netural landscape models, such as QRULE, it is impossible to fully separate the effects of habitat area and configuration. As a result, our analysis shows a high degree of confounding between habitat area and configuration. Most of the configuration metrics we tested have larger marginal and independent explanatory power than does habitat area (consistent with our expectation). However, most explained variance is shared between habitat area and configuration, meaning that it is not possible to formally ascertain the true relative effect. In cases of such confounding the magnitudes of marginal and independent explanatory power are typically used, which in our case suggests that configuration is has more influence on genetic differentiation than does habitat area.

An additional limitation of this study is that we chose a fixed population size across all simulations. The reason for this was to control for the effects of differential effective population size on the rate of genetic drift. By holding population size constant we eliminated the effects of differential rates of genetic drift on the strength of genetic differentiation. However, this also means that our study is unable to fully explore the interactions of variable population size and density with habitat area and configuration in influencing genetic differentiation. This is a topic for future research. However, given our goal of quantifying the relative effects of habitat area and configuration on genetic differentiation, we feel it was appropriate to control for the effects of variable population size.

A final limitation of our approach is how we quantify genetic differentiation. Our study, like others before it (Cushman and Landguth 2010; Landguth and Cushman 2010; Landguth et al. 2010), used the strength of the partial Mantel correlation between

genetic distance and cost distance, partialling out geographic distance, as our measure of the strength of genetic differentiation. The strength of the partial Mantel correlation is a good indicator of how closely genetic differentiation varies as a function of cost distance. Observing increased correlation between genetic distance and cost distance in landscapes with high fragmentation compared to those with low fragmentation is an indication that genetic differentiation is more strongly related to landscape structure when landscapes are fragmented than when they are not. However, the strength of the partial Mantel correlation does not describe the degree of genetic differentiation as a function of cost distance or the extent of genetic correlation. Wasserman et al. (2010) found that genetic neighborhood size is reduced in fragmented landscapes resulting in a shortening of the range of significant genetic autocorrelation and a steepening of the rate of genetic change as a function of cost distance. It would be interesting to compare our results which show that the strength of correlation between genetic distance and cost distance increases with habitat fragmentation, with analysis of how the rate of genetic differentiation and the range of genetic autocorrelation varies with habitat fragmentation. This is an interesting topic for future research.

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